Somatic Cell Counts in Bovine Milk

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SUMMARY

Factors which influence somatic cell counts in bovine milk are reviewed and guidelines for their interpretation are presented. It is suggested that the thresholds of 300 000 and 250 000 cells/mL be used to identify infected quarters and cows respectively. However, it is stressed that somatic cell counts are general indicators of udder health which are subject to the influence of many factors. Therefore the evaluation of several successive counts is preferable to the interpretation of an individual count.

Relationships between somatic cell counts and both milk production and milk composition are discussed. Subclinical mastitis reduces milk quality and decreases yield although the relationship between production loss and somatic cell count requires clarification. Finally the availability of somatic cell counting programs in Canada is presented.

RÉSUMÉ

La numération des cellules somatiques dans le lait des vaches

Cet article présente les facteurs qui influencent la numération des cellules somatiques dans le lait des vaches, ainsi que les directives propres à son interprétation. On suggère d'utiliser un seuil respectif de 300 000 et 250 000 cellules/mL pour identifier les quartiers et les vaches infectés. On insiste cependant sur le fait que la numération des cellules somatiques représente un indice général de l'état de santé du pis et qu'elle subit l'influence de plusieurs facteurs. L'évaluation de plusieurs numérations successives est par conséquent préférable à l'interprétation d'une seule.

Les auteurs commentent les rapports qui existent entre la numération des cellules somatiques et la production, ainsi que la composition du lait. La mammite asymptomatique altère la qualité du lait et en diminue la sécrétion, bien que la relation entre la baisse de production et la numération des cellules somatiques demande une clarification. Ils terminent en signalant la disponibilité de programmes canadiens de numération de cellules somatiques.

INTRODUCTION

Mastitis continues to be one of the most costly diseases of the dairy industry. In a recent (1976) survey the annual loss attributed to bovine mastitis in the U.S.A. was estimated to be U.S. $\$1.3 \times 10^9$ (5). Approximately 69% of this loss (U.S. \$81.32/cow) was attributed to reduced milk production resulting from subclinical mastitis, 18% (U.S. \$20.99/cow) due to the treatment of clinical cases, and the remaining 13% (U.S. \$15.04) due to losses incurred in replacing cattle.

One of the techniques used to monitor the level or occurrence of subclinical mastitis in herds or individual cows or quarters is to determine the somatic cell count (SCC) of milk samples. Indirect methods for doing this, such as the California Mastitis Test (CMT) and Wisconsin Mastitis Test (WMT) have been available for some time, as has the dirct microscopic somatic cell counting procedure. More recently, automated devices for rapidly determining the SCC of milk samples have become available. The two most commonly used are the Coulter Milk Cell Counter,[1] which counts particles as they flow through an electric field, and the Fossomatic,[2] which stains cells with a fluorescent dye and then counts the number of fluorescing particles. Both devices are capable of rapid inexpensive determination of the SCC in large numbers of samples. Details of the procedures used by each have been presented elsewhere (29) and will not be discussed further in this paper.

The latter technological advances have given rise to programs for the routine screening of quarter, cow (composite) and herd (bulk tank) milk samples. The purpose of this paper is to outline factors which may affect the SCC and to present some guidelines for their interpretation.

FACTORS AFFECTING SOMATIC CELL COUNTS

The ability to correctly interpret somatic cell counts depends on an understanding of the factors which may affect them. These factors may exert their influence at the quarter, cow or herd level.

Infection Status

The most important factor affecting the somatic cell count of the milk from an individual quarter, and consequently the cow and the herd, is the infection status of the quarter. In comparison, other factors have only a minor effect (55). Organisms colonizing the mammary gland may be divided into one group, referred to either as minor pathogens or commensals (e.g. *Corynebacterium bovis* or coagulase negative staphylococci) and a second group containing the major pathogens (streptococci spp., *Staphylococcus aureus* and coliforms being the most common). The somatic cell counts of milk from uninfected quarters have averaged 260 000 cells/mL in quarters with no previous history of mastitis and 600 000 cells/mL in quarters with a previous history of infection, with a resultant overall average for uninfected quarters of 314 000 cells/mL

[1]Coulter Electronics Ltd., Hialeah, Florida.
[2]Fossomatic, Foss Electric, Hillerod, Denmark.

(62). Cell counts in composite samples taken from cows with all four quarters free of infection have been reported to average from 113 000 to 251 000 cells/mL depending on the cow's age (18). Other authors (16,40,55) have reported averages of 170 000 and 214 000 (arithmetic averages) and 106 000 cells/mL (geometric mean). Cows harboring commensals have been reported to have somatic cell counts in composite samples that average from 190 000 to 519 000 cells/mL, depending on the cow's age (18), and an average of 227 000 cells/mL has been reported when all age groups were considered (55). Cows harboring major pathogens produce, on average, cell counts over 600 000 cells/mL (18,41,55,62) although a geometric mean of 492 000 cells/mL has been noted (16). Some variation in the cellular response elicited by various major pathogens has been demonstrated (55,62) but it does not appear possible to differentiate amongst the major mastitis pathogens on the basis of somatic cell count alone.

Number of Quarters Infected

The concentration of somatic cells in a composite (cow) milk sample is a function of the individual counts of the four quarters and their respective milk production. This fact becomes of major importance when the objective of a mastitis detection program, based on composite samples, is to detect and classify as infected, cows which have subclinical infection in one (or more) quarters. An approximate doubling of the SCC has been reported with each additional quarter that was infected (41). Using a total and differential SCC, the ability to correctly classify cows as infected or uninfected has risen from 77.9% to 92.7% as the number of quarters infected rose from one to four (37). Therefore, the dilution of high cell count milk from infected quarters with low cell count milk from uninfected quarters is an important consideration in the interpretation of composite sample cell counts.

Age

Many authors (3,4,13,21,55,60) have reported an increase in the cellular content of cows milk with increasing age. This increase is primarily due to an increased prevalence of infection in older cows and is not due to any large increase due to age *per se* (36,51). Examining only mastitis free cows some have found no increase with age (17), while others (18,41,61) have reported a slight increase. The latter reported increase may be due to a higher prevalence of permanent glandular damage from resolved infections in older cows.

It has been found however, that older cows have a greater cellular response to both minor and major pathogens (18,36). This latter finding has been attributed to a number of things, including more quarters being infected, more extensive tissue damage in long standing infections, and a greater cellular response in quarters that have been previously infected (18,62).

Stage of Lactation

Somatic cell counts have been found to be elevated immediately after calving, regardless of whether the cow is infected or not. The elevation in count has been variously reported to last for five days (51) to two weeks (12,41) and consequently elevated cell counts during the first two weeks of lactation must be interpreted with caution. Throughout the remainder of the lactation, somatic cell counts have been reported to increase as the lactation progresses (3,4,55). However, as with the effect of age, this is not primarily a physiological phenomenon but instead results from an increasing prevalence of subclinical infections with time (51,65). In studies where observations are restricted to uninfected cows, it was found that no rise in SCC throughout lactation occurred (18,41), nor that daily milk yield affected the somatic cell count (17). Whether or not there is a rise in the SCC of milk from uninfected cows immediately before drying off is debated. Some have not found a rise at drying off (17), while others have, although only after milk production had dropped below 4 kg/day (6). It would seem that if there is a rise at the end of lactation it only occurs immediately before drying off.

Season

In general, somatic cell counts are lowest during the winter and highest during the summer with peak levels usually being reported in July and August (elevated counts have been reported from April through to October) (6,7,35,36,42,43,64). The elevated counts of summer are reported to persist after the temperature humidity index starts to decline and this phenomenon has been called the "summer carry over" effect (64). The increase during the summer does not appear to be entirely due to elevated temperatures because attempts to reproduce the effect by putting cows in environmentally controlled chambers and increasing the temperature have not reproduced the same effect (47,53,64). During the summer in Scandinavia, cows on pasture (cooler temperatures) have had higher cell counts than cows confined to the barn (warmer temperatures), and the increase in cows on pasture was primarily seen in noninfected quarters (56).

Stress

Cows maintained in groups in loose housing may produce higher somatic cell counts when the groups are mixed. Mixing groups has increased the bulk tank SCC from approximately 175 000 to a peak of 420 000 cells/mL four days after mixing (31). However, no difference was found in weekly quarter somatic cell counts following the mixing of groups (2). It has been reported that there is no increase in SCC associated with cows being in estrus (25).

Attempts to artificially increase somatic cell counts by injecting cows with either corticosteroids or adrenocorticotropic hormone have met with mixed results. Some authors (63,64) report increases in the SCC while others (10,46) have found no change. Stress induced by isolating individual cows in a paddock and/or chasing them with a dog has increased the SCC with the greatest increases being found in cows with a previous history of mastitis (66).

Diurnal Variation

Although somatic cells can be detected in milk samples from all quarters of all cows, there is variation in the level produced throughout the day and from one day to the next. Diurnal fluctuation of the SCC has been reported by many authors (11,14,17,57,59,67). Cell counts are reported to be highest in the strippings or immediately after milking with these levels persisting for up to four hours before gradually declining to their lowest level which occurs immediately before the following milking (11,57,67). The magnitude of the increase from the lowest to the highest level has been reported to be as much as 70 fold (67). However, the correlation between cell counts in a foremilk sample and a total representative sample has been reported to be high ($r = 0.86$) (51). Therefore either of these types of samples are acceptable for routine use (11,51). This diurnal fluctuation has important consequences for anyone collecting milk samples at any time other than immediately before or throughout a normal milking.

Cell counts have also been reported to be higher in samples collected at the evening in comparison to the morning milking (14,17,59). This difference has been reported to be as high as 20% (59), but it presumably is a function of the time interval between the two milkings.

Day to Day Variation

Day to day variation in cell counts has been investigated in quarter, cow (composite) and herd (bulk tank) samples. Fluctuations in individual quarter samples from uninfected cows have run in parallel, suggesting physiological factors acting at the cow level (12). The average coefficient of variation in composite samples taken repeatedly over a short period of time has been found to be approximately 30-35% (11,59) whereas, over a whole lactation, the coefficient of variation has ranged from 59 to 301% (17). The latter authors have suggested that the periodic large increases in cell count that they detected in the absence of mammary pathogens may in fact have been due to infection with pathogens which were eliminated before they were isolated, or alternatively may have been due to stress or trauma. These data suggest that it is advantageous to sample cows or quarters several times throughout a lactation and sample at least five times during a lactation has been recommended (9).

The coefficient of variation in daily bulk tank counts has been reported variously to be 24% (65) and 23% (9). For individual herds the coefficient of variation in monthly bulk tank counts has been reported to range from 4 to 46% (21).

Technical Aspects

It has been shown that the method of transportation and storage of milk samples, as well as the method used to count the somatic cells, all have an influence on the resultant counts. These factors have been reviewed extensively elsewhere (24,28,44,45,58) and hence will not be discussed here except to stress the importance of consistency in the techniques used to handle and process samples in any somatic cell counting program.

Management

Mastitis control procedures primarily exert their influence on somatic cell counts at the herd level by influencing the quarter infection rate (40). In this regard, a number of studies have investigated associations between various control procedures and somatic cell counts in herds. The regular use of teat dip has consistently been associated with lower somatic cell counts (6,22,26,38,39,50,55). The extent of the effect of dry cow therapy on SCC appears to depend on how it is used. In one study lower bulk tank cell levels were associated with complete or "blanket" dry cow therapy in comparison to selective therapy (38), while in others (6,55) selective dry cow therapy was associated with lower SCC levels than complete therapy. The highest SCC were found in farms which did not use teat dip but which used complete dry cow therapy, and the lowest SCC in farms which used teat dip in conjunction with dry cow therapy (6). Dry cow therapy has reduced cell levels at the start of the subsequent lactation but teat dipping (or spraying) was required to maintain the advantage (27). The lowest cell counts have been reported to be in herds which adopted a full control program of teat dipping, blanket dry cow therapy and annual milking machine maintenance (8). It would appear that dry cow therapy is of most benefit when used in conjunction with other control procedures, particularly teat dipping. The use of individual towels has been associated with lowering cell levels (22,55) as has the design of the milking systems with the highest counts being reported in pipeline systems and the lowest in parlors (6,55).

TABLE I
RELATIONSHIP BETWEEN SOMATIC CELL COUNTS AND CMT REACTIONS

CMT	SCC/mL
—	0 - 200 000
Trace	150 000 - 400 000
1	300 000 - 1 000 000
2	700 000 - 2 000 000
3	> 2 000 000

INTERPRETATION OF SOMATIC CELL COUNTS

Somatic cell counts can cover a wide range of values. Counts as low as 7 000 cells/mL have been reported (61) and there is no theoretical maximum. However, when cell counts exceed 10×10^6 cells/mL the infection is likely to show clinical signs. Table I presents the approximate relationship between the CMT and the somatic cell count.

Interpretation of a SCC will depend on whether it is from a quarter, cow or bulk tank sample and as well involves consideration of several other aspects. First, if the cell counts are to be used to classify a unit (quarter, or cow) as positive or negative for mastitis, then no matter what threshold is chosen to divide the negative from the positive units there will always be some units incorrectly classified. With composite samples, for example, at any chosen threshold it is almost certain that some infected cows will have cell counts lower than the threshold, and will therefore be incorrectly classified as negative (or uninfected). Conversely, some uninfected cows will exceed the theshold and will therefore be incorrectly classifed as positive (or infected). The objective of interpretation is to minimize the rate of misclassification. Second, cell counts are merely a reflection of udder damage due to a variety of possible causes and as such they indicate more about the state of health of the udder than simply whether or not a pathogen is present. Finally, it should be stressed that the evaluation of herd averages and trends may be of more benefit than the evaluation of an individual quarter, cow or bulk tank count.

Quarter Samples

Both 400 000 and 500 000 cells/mL have been evaluated as possible thresholds, for classifying a quarter as being infected but both resulted in a high false negative rate (i.e. too many cases of subclinical mastitis were missed) (52). It has been recommended that the threshold be set at 300 000 cells/mL (34). Secretory disturbances, including declining milk yield, have been reported to start once cell counts exceed 100-150 000 cells/mL, and the probability of isolating a major pathogen is increased with counts above 200 000 cells/mL (51). On the other hand, cell counts averaging 600 000 cells/mL have been seen in cows which were currently uninfected but that had a previous history of mastitis (62). Until further work is reported, the use of a threshold of 300 000 cells/mL appears to be most reasonable for dividing quarters into uninfected and infected (with a major pathogen) categories.

Cow (Composite) Samples

When interpreting cell counts from composite samples it is important to consider the dilution effect that milk from normal quarters has on elevated counts from infected quarters. In general, negative cows had somatic cell counts less than 100 000 cells/mL while cows infected with minor pathogens (commensals) generally fell in the 100-300 000 cells/mL range (18). Production starts to decline once cell counts have risen above 100 000 cells/mL. It has been suggested that an appropriate threshold is 228 000 cells/mL and at that threshold it is possible to classify 85.8% of cows correctly (16). This success rate in classification compares favorably with an earlier work where both total and differential cell counts were used to correctly classify 79.4% of all cows (37). It has been suggested that cell counts under 500 000 cells/mL are normal (55), but in view of other work this value seems high. If the aim of a cell counting program is to detect cows which may only have subclinical mastitis in one quarter, then the use of a threshold, to divide uninfected cows from infected cows, of approximately 250 000 cells/mL appears reasonable.

If the average cell count for all cows in a herd is to be calculated then it has been suggested that a logarithmic transformation of the counts be made before averaging (14,59). This is equivalent to calculating a geometric mean and it has the desirable effect of preventing a few cows with exceptionally high counts from having an unduly large effect on the herd average. The logarithmic transformation of somatic cell counts has also been demonstrated to be optimal for the purposes of statistical analysis (1).

Herd (Bulk Tank) Samples

When interpreting bulk tank SCC it is important to remember that elevation of the count may result from a few cows having exceptionally high cell counts or from a general elevation of counts in many of the cows in the herd. The effect of one or two cows with extremely high somatic cell counts is particularly noticeable in small herds (55). In addition to this, bulk tank counts do not provide any information about which cows are affected. Never the less, they can serve as a useful indicator to alert dairymen to problems in the herd and also increase the producer's general awareness of subclinical mastitis.

Attempts to predict herd quarter infection rates from bulk tank counts have met with mixed success due to the fact that bulk tank counts are a function of both the quarter infection rate and the severity of those infections (51,61,65). Correlations between a single bulk tank SCC and quarter infection rates (QIR) have been reported variously to be r = 0.583 (51), r = 0.5 (50) and r = 0.5 to 0.6 (65). However, it has been suggested that improvement on this can be obtained by using either a three or six month rolling average. In one study it was only possible to correctly classify a herd as having a low (< 10%), medium (10-25%), or high (> 25%), QIR 45.5% of the time if only one bulk tank sample was used, but it was possible to increase this to 80% based on six previous monthly samples (37). Over a six month period the average bulk tank counts in low QIR herds ranged from 112 800 to 480 300 cells/mL while in high QIR herds they ranged from 609 400 to 729 700 cells/mL with medium QIR herds falling between the latter two groups.

Correlations between bulk tank SCC and the average (geometric mean) of all individual cows or quarters have been reported to be r = 0.83 (for cows) (51) and r = 0.89 (for quarters (48). These results would tend to indicate that although individual bulk tank counts may be of limited value in predicting the prevalence of bacterial infections in the herd, they do give a good indication of the general state of udder health. In general it appears that bulk tank counts under 250 000 cells/mL indicate a good level of udder health and that counts over 500 000 cells/mL indicate a definite herd problem with subclinical mastitis.

SOMATIC CELL COUNTS, MILK PRODUCTION AND MILK COMPOSITION

Production Losses

Losses in milk production attributable to mastitis were reviewed, (30) with losses at the quarter level being found to range from 9% to 43.3% of potential milk production. Table II shows the relationship between various CMT reactions in quarter samples and percentage milk production loss as reported by several authors (15,20,49). (One of these authors (15), and another cited in this section (32), reported losses on an absolute scale (pounds/day or gallons/year). For comparison purposes these losses have been converted to percentages based on an average production of 14 000 lb/cow/year).

At the quarter level, yields have been reported to start declining at 100 000 cells/mL (51), 200 000 cells/mL (33), and 500 00 cells/mL (55). However, the second study (33) stated that the decline was not significant until 500 000 cells/mL. Another study (62) reported the relationship between the SCC and production loss

TABLE II
PERCENTAGE MILK PRODUCTION LOSS ASSOCIATED WITH VARIOUS CMT REACTIONS IN QUARTER SAMPLES

CMT	% Production Loss		
T	8.4	9.0	2.8
1	19.0	19.5	11.4
2	33.8	31.8	25.6
3	50.0	43.4	45.5
Reference Number	15[a]	20	49

[a]Actual values reported were 0.96, 2.18, 3.88, and 5.74 lb/quarter/day

to be quadratic, as opposed to linear, but reported production loss at the quarter level to be approximately 7.5% for each 1 000 000 cells/mL increase in quarters with cell counts less than 3 000 000 cells/mL.

Losses at the cow level have been reported to be 6%, 10%, 16% and 24.5% for CMT reactions respectively of T, 1, 2 and 3 (23). The senior author (unpublished data) found an overall loss in milk production of 12.5% per increase of 1 000 000 cells/mL in composite samples. However, the relationship was not linear with the rate of loss being greatest in the 200 000 to 1 500 000 cells/mL range. A loss of 1.4 litres/cow/day has been associated with an increase of 1 000 000 cells/mL in composite milk samples (comparing cows within herds) (21). The loss associated with a similar increase in SCC of bulk tank samples (comparing herds) was approximately twice as large (2.7 litres/cow/day). The authors attributed the difference to other management deficiencies which accompanied poor mastitis control. Losses have been reported to be respectively 42, 74, 169, and 197 gallons/cow/year for bulk tank cell count ranges of 250-499 000, 500-749 000, 750-999 000, and greater than 1 000 000 cells/mL (32). (It is estimated that these would represent production losses respectively of 3.0%, 5.3%, 12.1%, and 14.1%).

In general, elevated somatic cell counts are indicative of production losses due to subclinical mastitis. It appears that although there is considerable variation in the literature as to the magnitude of the loss, it may frequently exceed 20% of a cow's potential production. Further research is required to elaborate the exact relationships between SCC and production losses at the quarter, cow and herd level. However, it must be remembered that the reduction in milk production is only one of the many losses associated with mastitis and that losses due to other factors such as, discarded milk, drug costs, veterinary fees, extra labour, increased cow replacement costs and loss of genetic potential should also be considered (15).

Milk Composition

It has been demonstrated that subclinical mastitis results in increased levels of blood constituents in milk and decreased levels of the components of mammary origin. For example high cell count milk has lower fat, solids-not-fat and lactose levels and increased levels of sodium, chloride and free fatty acids than low cell count milk (19,32,33,54). The total protein content of milk does not seem to change significantly, however a decrease in the level of casein and an increase in the levels of albumin and immunoglobulins have been reported (19,53). These shifts in composition have important consequences to the dairy industry. For example, the yield of cheese from high cell count milk has been reported to be lower than it is from low cell count milk. A loss of 0.31 lb of cheese per 100 lb of milk in bulk tank milk with a cell count of 640 000 cells/mL has been reported (19). In addition, the higher levels of free fatty acids in high cell count milk may produce a "rancid" flavor. As a result of this, some dairies find it economically advantageous to pay a premium for high quality, low cell count milk.

TABLE III
SOMATIC CELL COUNTING PROGRAMS IN CANADA — 1981

	BC	Alta	Sask	Man	Ont	PQ	NB	NS	PEI	Nfld
Bulk tank samples	yes	yes	yes	yes	yes[a]	yes	yes	yes	yes[a]	no
Individual cow samples	no	yes[a]	yes	no	yes	yes	yes	yes	no	no

[a]Program under development

SOMATIC CELL COUNTING PROGRAMS IN CANADA

To the best of the authors knowledge, Table III indicates the current availability and use of somatic cell counting in Canada. At the time of writing, there were several programs which were under development and scheduled to be operational in 1981. These programs have been included in Table III.

In those provinces in which cell counting is available on an individual cow basis it is usually provided through the provincial D.H.I. (or D.H.A.S.) organization although the service is often available to other producers (i.e. R.O.P. herds) as well.

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BOOK REVIEW

Advances in Parasitology, Volume 18. Edited by W.H.R. Lumsden, R. Muller and J.R. Baker. Published by Academic Press Inc. (London) Ltd., London, England. 1980. 364 pages. Price $48.50.

The 18th volume of this prestigious series comprises of six reviews. Three reviews are concerned with protozoa, two with ticks and one with helminths. The editors indicate that their policy should be to interpret parasitology in its widest sense while trying to select subjects for review of which our knowledge had advanced significantly up to the time of publication and this they do in this volume.

The review on helminths is Part III on the Seasonal Occurrence of Helminths in Freshwater Fishes by J.C. Chubb of the University of Liverpool, England and deals with larval cestoda and nematoda. Part I dealing with Monogenea and Part II on Trematoda previously were published in Advances of Parasitology Volume 15 and 17 respectively.

The life cycles of many larval cestodes and nematodes, some of which are of economic or of public health significance, of example, *Triaenophorus crassus, Diphyllobothrium dendriticum, Ligula intestinalis, Digramma interrupta, Eustrongylides* spp and *Diphyllobothrium latum* are briefly summarized followed by the seasonal infestations related to the major climatic zones of the world. The author concludes his review by arranging and summarizing the considerable information gleaned from about 300 references under such headings as incidence and intensity of occurrence, principal and auxiliary hosts, invasion of fishes by larvae, growth of larvae in fishes, microphological differences, longevity, mortality of heavily infected fishes, sporadic population changes, immunity seasonal studies in world climate zones, a hypothesis for seasonal occurrence and experimental studies.

The second review in this volume is a most comprehensive paper on Rumen Ciliate Protozoa by G.S. Coleman from the Agricultural Research Council Institute of Animal Physiology, Cambridge, England. This review includes consideration of the apparent role of protozoa in ruminant growth, metabolism and disease followed by brief accounts of cultivation and structure and detailed descriptions of physiology and biochemistry of individual species. There is a concluding summation on the role played by protozoa in normal ruminants. Large animal practitioners involved with the nutrition and metabolic diseases of ruminants would surely find this review both informative and rewarding.

The other two reviews on protozoa are concerned with two prospects of trypansomiasis. In the first of these two reviews, W.C. Gibson, T.F. de C. Marshall and D.G. Godfrey of the London School of Hygiene and Tropical Medicine present a new approach to the epidemiology and taxonomy of trypanosomes of the subgenus *Trypanozoon* by a numerical analysis of enzyme polymorphism. They discuss and compare enzyme electrophoresis with other methods of classification of trypansomes and attempt to correlate some of the electrophoretic results with available epidemiological data. Anyone working in the fascinating jungle of trypanosome taxomony will find this review refreshing and most worthwhile.

The second of these two reviews on trypanosomes deals with immunity to *Trypanosoma cruzi* by Z. Brewer of Brazil. This is a fascinating review on Chagas' disease, which affects millions of people in Latin American countries, and apparently for which there are no cures or methods of prevention. This review touches on such interesting aspects of the disease as natural immunity, effects of immunosuppressors, immunodepression in the course of the disease, evasion of the immune response by the parasite, auto-immune reactions and vaccination.

The final two reviews are on ticks. P. Willadsen of CSIRO, Indooroopilly, Queensland, Australia reviews the relatively scanty literature on immunity to ticks outlining the nature and different types of immunological responses concluding with a discussion of artificial immunization and the nature of tick antigens.

K.C. Binnington and D.H. Kemp also of CSIRO, Indooroopilly, Australia present a review of the role of the tick salivary glands in feeding and disease transmission. The authors present a good discussion of salivary gland function during attachment and feeding with changes in salivary gland morphology. They conclude with discussions on toxicosis and disease transmission particularly with regard to the function of the salivary gland and salivary gland secretions in these phenomena.

H.J. Smith.